Undersea Biomedical Research, Vol. 12, No. 1, March 1985

CLINICAL COMMUNICATION

Damage to the middle ear and the inner ear in underwater divers

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Money KE, Buckingham IP, Calder IM, Johnson WH, King JD, Landolt JP, Laufer J, Ludman H. Damage to the middle ear and the inner ear in underwater divers. Undersea Biomed Res 1985; 12(1): 77–84.—Postmortem human tissue from recently deceased divers was processed histologically to assess any inner and middle ear damage that could have resulted from the effects of pressure during diving. The following new findings are particularly noteworthy. In one diver, ascent while breath holding resulted in the rupture of the ear drum and blood in the middle ear, in addition to pulmonary barotrauma. In a second diver, following inner ear decompression sickness, new bone growth, similar to that described earlier in experimental studies with the squirrel monkey, was observed in the arms of one of the semicircular canals. These observations are further confirmation that otologic disorders can be a serious threat to divers.

inner ear	decompression sickness
middle ear	barotrauma
semicircular canals	pulmonary barotrauma
cochlea	divers

The direct action of pressure changes that are encountered in diving can cause damage to the ear in at least three distinctly different ways: (a) middle ear barotrauma of descent, (b) inner ear barotrauma, and (c) middle ear barotrauma as a manifestation of pulmonary barotrauma of ascent.

Middle ear barotrauma of descent occurs when there is a failure to equalize middle ear and ambient pressures by means of the eustachian tube. If that occurs, a relatively low pressure in the middle ear during descent (1), if large enough, can cause bleeding into the middle ear and even rupturing of the ear drum.

In attempting to raise the pressure in the middle ear during descent, clearing the ears with a Valsalva maneuver, if attempted too vigorously, can result in breaking the round window

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membrane and leaking of perilymph into the middle ear. One postulated mechanism (2) to explain this damage includes raising the intracranial pressure by the Valsalva maneuver, the transmission of this pressure to the perilymph space, and a progressive bulging of the round window membrane into the middle ear until a rip in the membrane allows perilymph to escape. Another postulated mechanism (3, 4) includes the sudden success of a forceful Valsalva maneuver: the inward-bulging ear drum suddenly bulges outward, pulling the stapes outward and "blowing in" the round window membrane with resulting rupture of the round window membrane. Regardless of the mechanism of damage, inner ear barotrauma (3) results in perilymph leak into the middle ear (5), and the diver experiences some or all of dizziness, tinnitus, and hearing loss. In some divers, bed rest with the head elevated for several days allows healing to take place, but in other cases surgical repair of the round (or oval) window is required to prevent permanent loss of hearing (4–7).

During ascent from a dive, the eustachian tube normally opens without difficulty and large pressure differences across the ear drum do not occur. However, if the breath is held during ascent (a mistake that is frequently fatal because of pulmonary barotrauma), the reduction of pressure outside the ear drum could, in theory, result in the bulging of the ear drum outward, causing rupture of the ear drum with concomitant bleeding into the middle ear.

Unrelated to the damage that is caused directly by these pressure changes is the indirect damage, i.e., by means of decompression sickness of the inner ear. When a dive is deep and prolonged, excessive quantities of nitrogen or other inert gases are dissolved in the body tissues. If the ascent is too rapid, these gases will come out of solution and form bubbles that can cause extensive damage. Such decompression damage to the inner ear is one of the most common kinds of bends associated with deep diving (8), and it creates hearing losses and/or dizziness in divers (9, 10). The damage is usually unilateral and can be reversed with prompt, standard recompression procedures (9, 10), including hyperbaric oxygen therapy (11), but sometimes the result is permanent damage (even a "dead ear"). In experimental animals, decompression damage to the inner ear includes bleeding into the perilymph and endolymph fluid spaces of both the cochlea and the vestibular apparatus (12–15), small breaks in the temporal bone (16), and invasion of the semicircular canals by new bone growth (16, 17).

The present study used postmortem human material to determine whether the clinical manifestations of damage to the ear in diving are consistent with the observed pathology, and whether the pathological findings in experimental animals have their counterparts in human material.

METHODS

The study material includes the temporal bones of three divers; one who had died from pulmonary barotrauma because of breath holding during ascent (to see whether there was bleeding in the middle ear space), one who had a history of inner ear decompression sickness and who died about 2 mo. later from an unrelated nondiving cause, and one who died underwater for reasons unknown. The temporal bones were sectioned serially for histological study using the celloidin method of Igarashi (18).

RESULTS AND DISCUSSSION

Pulmonary barotrauma of ascent

It was found that bleeding into the middle ear and rupture of the ear drum do occur in association with the pulmonary barotrauma that results from ascent while holding the breath.

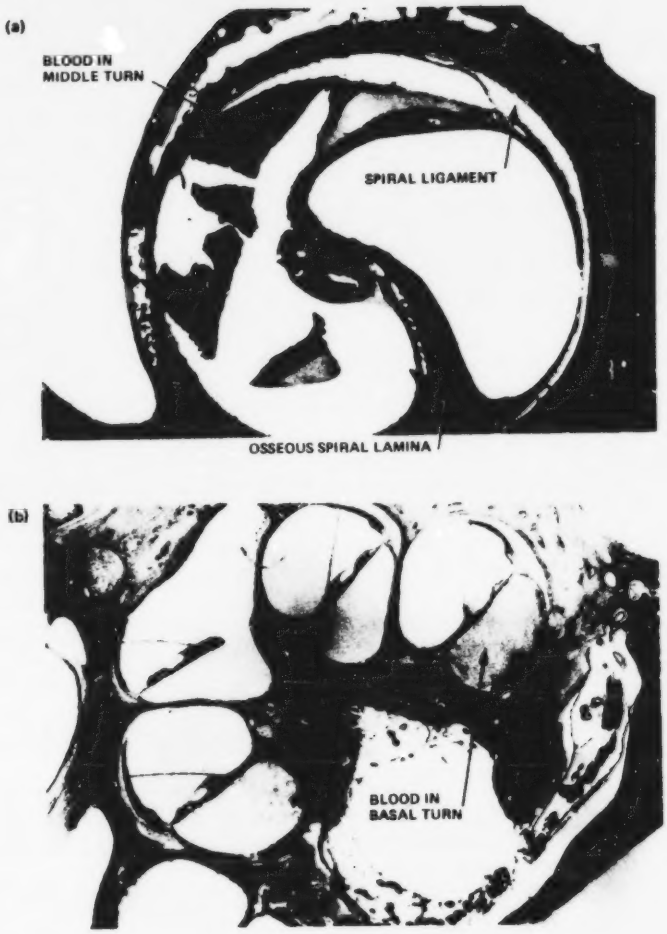

Fig. 1. Blood in the cochlea of a squirrel monkey and the cochlea of a human diver, following decompression sickness. *(a)* Blood in the middle turn of the cochlea in the diver; *(b)* Blood in the basal turn of the cochlea in the monkey.

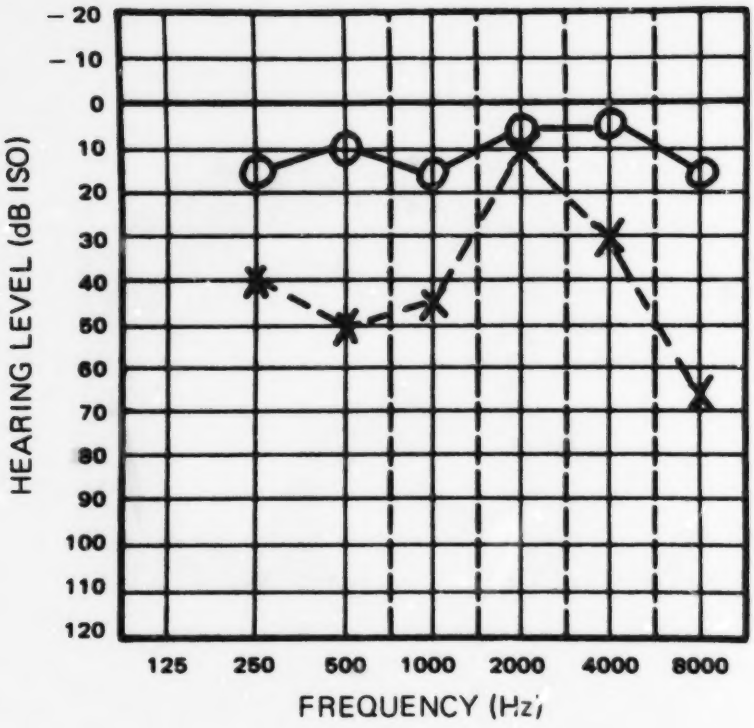

Fig. 2. Audiograms of diver following decompression sickness. *Open circles* indicate near-normal levels in the right ear; *X*'s indicate the very large hearing losses in the left ear. This diver also had complete loss of vestibular function in the left ear.

In the diver who died underwater from pulmonary barotrauma, both ear drums were ruptured and bleeding into the middle ear resulted in the blood appearing in the external ears. The amount of blood observed is much greater than the amount that could come from the tiny vessels supplying the tympanic membrane itself, and it seems likely that a direct pressure difference caused rupturing of vessels in the lining of the middle ear cavity.

Ascending with closed glottis is one of the most elementary and basic errors a diver can make, and it is therefore reasonable to think that such an error would usually occur only after a considerable provocation such as (a) local trapping or gross contamination of the breathing gas in the unsuspecting diver (for example, if the diver inhaled oil or water or vomitus or had his air trapped, he might panic and hold his breath while swimming for the surface); (b) decrease in cerebral competence (for example, a toxicosis); or (c) a disorientation (for example, in conditions of restricted visibility a vestibular malfunction could make the diver think he was swimming down when he was actually swimming up). Disorientation with erroneous perception of the down direction occurs readily underwater (10) and, at least in aircraft pilots, disorientation can be strongly compelling even when correct symbolic information about orientation is available.

Fig. 3. Sections across a normal *(top)* and an abnormal *(bottom)* semicircular canal from a human diver. This diver suffered decompression damage to his left ear and died of unrelated causes 56 d later. The pathological new bone growth in the *(bottom)* semicircular canal is typical of that produced in monkeys by decompression damage.

Inner ear decompression sickness

As in experimental animals (14), it was also found in human material that decompression sickness (resulting from bubbles of inert gases coming out of solution with decreasing pressures) can cause bleeding into the perilymph spaces of the cochlea. Figure 1 shows this phenomenon in a squirrel monkey that had been exposed to a simulated dive (17) and in a human diver. It is possible that this bleeding is associated with the hearing losses that sometimes occur with decompression sickness in human divers (9, 10) and in animals exposed to similar pressure schedules (19).

Fig. 4. Middle and inner ears, left *(a)* and right *(b)*, from a diver who died underwater from causes unknown. Left ear has the round window membrane ripped and the middle ear filled with blood; right ear is essentially normal.

One human diver suffered severe inner ear symptoms, hearing loss with tinnitus, and dizziness some 5 h after ascent from a dive to 100 meters of sea water (msw) for 19 min. The episode was clearly one of decompression sickness and it included some joint pain (another common result of inert gases forming bubbles in tissues). It was therefore entirely unrelated to the middle ear problems of diving; it was caused by bubbles of inert gas. Subsequently, his hearing loss in the left ear was documented clinically (Fig. 2) as was his complete loss of vestibular function in the left ear. Moreover, a variety of recompression treatments were employed at the time of the hit, none of which alleviated the condition. This diver died suddenly of unrelated causes, 56 d after the dive that caused his decompression damage. Figure 3 shows

the new bone growth that had started to invade one of the semicircular canals of his left ear. This peculiar and manifestly maladaptive bone growth has been shown to occur in squirrel monkeys as a result of decompression damage to the inner ear (17), and it is significant that decompression sickness can also provoke this bone growth in man.

In monkeys, new bone growth following decompression sickness seems to be initiated by rips in the endosteum or by tiny fractures (with displacement of small chips of bone) in the bony semicircular canals (16, 17). The inner ear will produce maladaptive new bone growth in response to a variety of stimuli, and in man such growth has destroyed the inner ear following trauma to the endosteum, inner ear hemorrhage, infection, or interference with the blood supply (20, 21).

Other findings

Figure 4 shows the middle ears and inner ears of a diver who died underwater from causes undetermined. In this diver, the round window membrane was ruptured on the left side and the middle ear was almost filled with blood. Blood precipitates were also found in the cochlea. On the right side, the round window membrane was intact, the middle ear contained only a little blood, and the cochlea was free of precipitates. The large amount of blood in the left middle ear suggests that the bleeding occurred under pressure (that is, while the heart was beating). The pressure required to rupture the round window membrane could have been produced by a violent Valsalva maneuver or by an ascent with a closed glottis. It is possible that the rupture of the round window membrane was followed by a vestibular malfunction, resulting in disorientation that contributed to the death of the diver.

CONCLUSIONS

Following decompression sickness of the inner ear, similarities were found in the histological pictures of the ear damage in man and in monkeys. The new bone growth in the semicircular canals, found here in man following inner ear decompression sickness, can be expected to attenuate the functions of the semicircular canals over the following several months, and such attenuation when observed clinically can be understood on the basis of events in the inner ear, without recourse to etiologies involving the central nervous system (22). Such maladaptive bone growth might also be responsible for the "late deficit" that can follow the more common kinds of fracture or other injuries of the temporal bone (Barber HO, Personal Communication).

Evidence was found that in addition to pulmonary barotrauma, ascent while breath holding can result in bleeding into the middle ear and rupture of the ear drum. This study also shows quite clearly that, if a death occurs underwater for unknown reasons, it would be prudent to examine carefully the inner ear and middle ear histologically to determine whether an air embolism preceded or precipitated the death.

Ms. Dawn Gardham typed this manuscript. This study was supported in part by the Office of Naval Research, USN, Grant N00014-77-G-0077. DCIEM Research Paper 85 P-09.

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Money KE, Buckingham IP, Calder IM, Johnson WH, King JD, Landolt JP, Laufer J, Ludman H. Lésions de l'oreille moyenne et de l'oreille interne chez des plongeurs sousmarins. Undersea Biomed Res 1985; 12(1):77–84.—Des tissus humains de plongeurs récemment décédés furent traités histologiquement afin de vérifier les lésions possibles de l'oreille interne et moyenne qui auraient pu

résulter des effets de la pression durant la plongée. Les découvertes nouvelles suivantes sont particulièrement remarquables: (a) chez un plongeur, la remontée en apnée produisit une rupture du tympan avec effusion de sang dans l'oreille moyenne, en plus de barotraumatisme pulmonaire; (b) dans un deuxième plongeur ayant subit une maladie de décompressin de l'oreille interne, une nouvelle croissance osseuse ectopique, semblable à celle décrite antérieurement dans des études expérimentales chez le sagouin (*Saimiri sciureus*), fut observée dans les bras de l'un des canaux semicirculaires. Ces observations sont une confirmation supplémentaire que les troubles otologiques peuvent être une menace sérieuse pour les plongeurs.

oreille interne
oreille moyenne
canaux semicirculaires
cochlée
maladie de décompression
barotraumatisme
barotraumatisme pulmonaire
plongeurs

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